

Periscope

BRAIN

(Vol. 27, 1904, No. 106, Summer.)

1. A Contribution to the Study of the Cortical Localization of Vision. A Case of Quadrantic Hemianopia with Pathological Examination. C. E. BEEVOR and JAMES COLLIER.
2. Some Cases of Family Disseminated Sclerosis. ERNEST S. REYNOLDS.
3. Fecal Vomiting and Reversed Peristalsis in Functional Nervous (Cerebral) Disease. A Summary of Cases and Conclusions. F. PARKES WEBER.
4. Intramedullary Abscess of the Spinal Cord. An Account of Three Cases. WILLIAM ALDEN TURNER and JAMES COLLIER.
5. Bilateral Loss of Postcentral Cortex, Apparently Congenital, in Adult. HUBERT M. TURNBULL.

1. *Cortical Localization of Vision.*—The authors have investigated a case of quadrantic hemianopia and present the results of a pathological examination with microscopical studies. The patient during the greater part of the time that he was under observation showed a partial blindness in the left upper quadrant in both visual fields, the fixation point escaping. This condition had remained constant for a period of nearly two years. An occlusive lesion of the right posterior calcarine artery had caused destruction of the cortex: (1) of right fusiform lobe posterior 2 cm.; (2) right lingual lobe from junction of calcarine and parietal occipital fissures to the pole of the hemisphere; (3) of the whole cortex in the depth of the calcarine fissure; (4) of the greater part of the inferior cuneal gyrus, small areas only at the anterior and posterior limits of the gyrus being free. The necrosis did not involve the optic radiation at any point. The only parts of the cortex of the occipital lobe which had escaped destruction were the upper two-thirds of the cuneus and the anterior and ventral portion of the fusiform lobe. The lower quadrants of the visual fields were entirely unaffected, and until it was pointed out to him the patient was unaware of the visual defect. The authors submit, therefore, that this case affords conclusive evidence that the cortex of the upper two-thirds of the cuneus is the primary visual center for the lower quadrants. The following inferences are drawn from the case: (1) The larger part of the lesion was situated below the calcarine fissure. The upper quadrant is chiefly, if not entirely, represented below the calcarine fissure. (2) The cortex lining the calcarine fissure was completely necrotic. The primary half vision center cannot, therefore, be limited to the calcarine cortex nor the macula exclusively represented in the anterior part of this region. (3) The portion of the half-vision center, as limited by von Monakow, which was not involved was the upper two-thirds of the cuneus. (4) The visual defect was blindness of both left and upper quadrants. The lower quadrants are represented to a great extent in the upper two-thirds of the cuneus. (5) The lower quadrants of the visual fields were not affected, and the patient was not himself aware of any visual defect.

2. *Family Disseminated Sclerosis.*—The authors put on record two instances in which this condition may be said to have affected more than one member of a family. In one family the father is suffering from melancholia, the first cousin of the father died of disseminated sclerosis. A sister of the mother suffered from melancholia. There were five children. The

eldest, female, died from melancholia; the second, female, had multiple sclerosis; the third has severe sciatica; the fourth is one of the cases reported; the fifth is well, and the sixth is the third case of multiple sclerosis reported. The second family consists of three daughters, M., C. and J. M. The eldest, 29 years old, suffers from advanced multiple sclerosis; C. is in good health, and J. M. is reported in the history as another case of multiple sclerosis. These patients are all suffering from irregular types of the disease.

3. *Reversed Peristalsis in Nervous Diseases.*—F. P. Weber presents an interesting summary of this condition, from which he draws the following conclusions: (1) Functional nervous vomiting, like the hemianesthesia, palsies and spasms of hysteria, must be regarded as due to an abnormal state of the cerebral cortex, and is just as much a symptom of functional brain disease as the vomiting in cases of cerebral tumor is of organic brain disease. (2) Fecal vomiting of functional nervous origin is merely a rare and extremely exaggerated form of ordinary hysterical vomiting. (3) The vomiting in functional brain disease may sometimes be more violent and severe than it ever is in organic cerebral disease, since fecal vomiting is scarcely known to occur in cases of cerebral tumor, etc. Some light is thrown on this point by the fact that a delusion is apt to be more stable and better "organized" in a monomaniac whose brain, could it be examined, would probably show no obvious change, than in a general paralytic whose brain is the seat of grave organic disease. (4) For the occurrence of fecal vomiting of functional nervous origin active intestinal antiperistalsis is absolutely necessary. But it is not certain that antiperistalsis necessarily always plays a part in the fecal vomiting known to surgeons as a symptom of organic intestinal obstruction (organic ileus). (5) The fecal vomit in organic obstruction of the bowel is seldom, if ever, more than "feculent"; that is to say, having the odor of feces without containing visible fecal particles or masses. Vomiting of formed feces in the absence of malingering and gastro-colic fistula practically only occurs in functional nervous cases. This may partly be accounted for by remembering that antiperistalsis, if it occurs at all, is likely to be more forcible when the muscular walls of the gut have not been previously weakened by overdistension or gross organic disease. (6) Hysterical malingering is, of course, apt to develop in the same (hysterical) class of patients in whom fecal vomiting occurs, and the possibility of genuine fecal vomiting occurring side by side with simulation must be kept in mind.

4. *Intramedullary Abscess of the Spinal Cord.*—Drs. Turner and Collier report on three cases of this rare condition following suppurative leptomeningitis which have come under their observation. The cases resembled each other closely, being cases of pressure paraplegia in which a transverse portion of the spinal cord was the seat of myelitis or necrosis from loss of blood. In all cases the suppuration commencing in the necrotic area spread the length of the spinal cord above and below. In the first case the paraplegia was slow in onset, and had been complete for several weeks when suppuration occurred. Though it is certain from a pathological examination that the suppuration occurred subsequent to the evascularization, nothing in the clinical history of the case seemed to indicate at what period suppuration commenced. The physical condition was feeble. Rigors did not occur, nor was the temperature above normal throughout. There was no alteration in the spinal symptoms indicative of the upward extension of the abscess. In the second case the paraplegia became complete in a few days after the first appearance of spinal symptoms, and this was evidently the result of extensive strangling of the spinal arteries by pachymeningitis in the lower half of the dorsal region. The patient lived a month after the onset of paraplegia. The advent of suppuration was presumably marked by the occurrence of pyrexia and rigors, which persisted until death, but by no other change in the clinical picture referable to the nervous sys-

tem. In the third case the onset of paraplegia was rapid. The presence of pyrexia during the time the patient was under observation, and the fact that the spinal symptoms progressed as if from a spreading lesion, made it probable that suppuration rapidly followed the occurrence of myelitis and that the spread of symptoms was evidence of its extension. That suppuration commenced subsequently to the occurrence of the myelitis is, we think, clearly proved by the fact that in the limited transverse area of the compression myelitis the spinal cord was completely necrotic, and at this point the abscess was least in evidence, tissue reaction being possible in the necrotic area; but above and below the lesion, where the abscess was conspicuous and extended for many inches in the length of the cord, there was no sign of such necrosis. The paper is profusely illustrated.

5. *Bilateral Loss of Postcentral Cortex*.—An extensive description of a bilateral loss of the postcentral cortex, apparently congenital, in an adult 24 years of age, who died from severe burns. Life history was obtained from the patient's mother and sister. Patient was delivered in an easy labor. As a baby she had several infantile convulsions, and two severe fits with unconsciousness, one as late as her seventh year. She frequently had fits of temper and would scream and strike at people. These attacks never had any of the earmarks of the psychic equivalents of epilepsy. She never had any attacks of paralysis or transitory weakness. She was late as a child in her speech, and was able to converse rationally, but she was distinctly undeveloped. She was practically blind and imbecile from birth. Her hearing was acute, and she understood the significance of words, particularly of the sounds connected with the preparation of food. Her sense of smell and taste was distinct. She could smile when pleased, but never laughed heartily. She had control of her sphincters. She could never dress herself, do her hair or blow her nose, and had been growing distinctly weaker. Examination showed complete loss of the postcentral cortex on both sides, which space was occupied by enormous cysts, each cyst being covered by arachnoid and representing attenuated portions of the cortex. On the left the cyst was an evolution from the thin cortex of the limbic lobe immediately below the notch representing the anterior limb of the calcarine fissure. Thus the thin cyst replaced all the parietal lobe save the ascending parietal and one-half of the supramarginal convolution, practically all the temporal lobe save the pole and the anterior two-thirds of the supra-temporal convolution and the temporal operculum, all the precuneus on the left and posterior two-thirds on the right, the whole occipital lobe, including the cuneus and all but a small fragment of the lingual lobule. The abnormalities found by microscopic examination consisted of a distension of the ventricles and destruction of a large area of the cortex accompanied by certain changes in the remaining cortex, in the corpus callosum, in the retro- and sub-lenticular portions of the capsule, and in the optic nerve and its central connections. The facts settled beyond dispute in the study of the interdependence of parts of the central nervous system justify the statement that the changes in the corpus callosum, the capsule and the optic nerve connections were undoubtedly secondary effects of the cortical destruction, and suggest a similar explanation of the abnormalities in the thick cortex. The physical deductions were of considerable interest, and well worth quoting in toto.: "Finally, from the life history of the patient it is possible to estimate the physiological effects of the cortical destruction, and to see how far these agree with the present views on cortical localization. *The olfactory sense* was present and played an unusually important part in the choice of food, replacing the lost sense of vision. That it was not interfered with by the lesion was to be expected, seeing that all those parts of the brain were intact which, from the study of comparative anatomy and such experimental and clinical evidence as have been obtained, have been associated with the sense of smell. Those parts are: the olfactory bulb and tract, the uncus, the hippocampal convolution,

hippocampus major, dentate convolution, nerves of Lancisi, peduncles of corpus callosum, fornix, fimbria, anterior commissure and the callosal convolution. *The sense of taste*, the cerebral center of which is probably in close association with the olfactory, was also present. *The neuromuscular mechanism*.—The limbs could be moved at will, and the sphincters were under voluntary control. There was, however, latterly weakness in muscular movements and inability to walk without support. That the motor area of the cortex—applying the term to the area containing the cells which give origin to the pyramidal projection fibers—was not interfered with was shown by the perfect condition of the genu and the posterior limb of the internal capsule and of the pyramidal tracts. In this connection it is interesting to recall the condition of the ascending parietal convolutions. Not only were these absolutely and relatively extremely small, but on microscopic examination it was seen that, where the cyst had origin from this convolution, the adjacent portion of the convolution was entirely devoid of nerve cells. That such a condition of the parietal convolutions was compatible with an anatomically perfect pyramidal tract is consistent with the observations of Sherrington and Grünbaum that in the anthropoid apes the postcentral convolutions are electrically inexcitable, and their injury is not followed by degeneration in the pyramidal tracts. No marked wasting was seen in the limbs and the anterior horn cells of the cervical and lumbar enlargements of the cord were healthy. It is reasonable, therefore, to suppose that the talipes equinovarus noticed in the necropsy was the result of long sitting in the tailor position and not of a spastic paralysis. It is difficult to understand why the patient, who had been able to walk spontaneously and unaided, lost that faculty; had the loss been accompanied or preceded by a fit or by a loss of muscular power it would have clearly pointed to a progression of the destructive lesion, but no loss of power could be noticed at the time and the behavior of the patient was such as to make it appear that there was a disinclination rather than an inability to walk. Her mother made no effort to overcome the disinclination, and it is possible that the power of spontaneous and unaided progression was ultimately lost through disuse. *Of muscular and tactile sense* little can be said. That she could lift and play with small objects, although blind, and could modify the force of her muscular efforts, for instance, using her hands to put food in her mouth, or striking violently when angry, pointed to the integrity of these senses in the arms at least. The sense of touch, however, did not seem to play an important part in her appreciation of the outside world. *As regards speech*, the neuromuscular mechanism of articulation was normal. The phonogenetic center must have been present, and this must have been in connection with the auditory center to enable her to speak. Not only did she recognize the sounds of certain words, but she associated certain facts with them, and could even associate sounds heard with words; thus, she would repeat "Annie is going to have some fish" on hearing fish being fried, so that she may be said to have possessed an auditory word center. The connection of the auditory word center and phonogenetic center, with the higher centers of intelligence and memory, seems to have been present, in that she could answer certain questions correctly in the affirmative or negative, and in that she could remember verbal promises. In considering the possibilities of her brain in this connection it must be remembered that no attempts were made to educate her. *Her hearing was acute*, and with the exception of the part played by the senses of smell and taste, her auditory sense seems to have been the link between herself and the outer world. It was by hearing and not touch that people were recognized; on hearing depended such powers of speech as she learned, and it was by hearing that she recognized what was going on around her, as shown, for instance, by her screams of anger when the furniture was moved preparatory to washing the floor, a proceeding associated in her mind with being disturbed from her usual seat.

(Vol. 27, 1904, No. 107, Autumn.)

1. On the Cells of the Spinal Ganglia and on the Relationship of Their Histological Structure to the Axonal Distribution. W. B. WARRINGTON.
2. On Certain Tremors in Organic Cerebral Lesions. GORDON HOLMES.
3. The Pathology of Infantile Paralysis (Acute Anterior Poliomyelitis). FREDERICK E. BATTEN.
4. So-Called Facial Hemihypertrophy. H. MACKAY.

1. *The Cells of the Spinal Ganglia.*—The authors report the results and conclusions reached by a series of experimental lesions on cats. They review briefly the work of Doglei and others, who have showed that spinal ganglia contain not only the usual cell with the T-shaped axon of Ranvier, but others with differently distributed axons. The second cervical ganglion, which receives afferent fibers from the skin and small muscles, the eleventh dorsal, which also receives afferent fibers from the viscera; the seventh lumbar, which receives afferent fibers from the large hamstring muscles and has no visceral connections, were selected and 1,000 cells from each carefully measured, showing wide variations in size. Whereas in the dorsal and cervical region considerable similarity was noted in the proportion of cells of different size, in the lumbar ganglion appear a number of larger cells, and the authors conclude that these represent the cells in connection with the sensory organs, the muscle spindle of the large hamstring muscle. They further endeavor, following the work of Lugaro, Orr and Rows, to study by means of experimental section of nerve fibers of different distribution the axonal reaction occurring in the cells of corresponding ganglia, and to classify the types of cells thus identified according to the system of Lugaro. Chromatolytic changes were found occurring in the obscure cell and in the large clear cell, but the cell of the large granules, termed by the authors the coarsely granular cell, changed only when the section was made just beyond the ganglion, and the cells measuring less than 25 mmm. never showed any chromatolytic change. The paper is profusely illustrated, and the authors sum up as follows: (1) The size of the cells in the ganglia is a function of the diameter of the nerve fiber, and the difference of appearance in the chromophile elements has a dynamical significance. These elements represent material which is used up by the essential trophoplasm, which presides over the nutrition of the nerve fiber. (2) At the region of the limb plexuses a number of large cells appear in the ganglia, which are the cells of origin of the fibers to sensory muscle structures. (3) Lugaro's classification is confirmed in the main. The cells met with are best described as (a) clear cells, (b) obscure cells, (c) coarsely granular cells, (d) smallest clear cells. (4) The large clear cell and the obscure cell give origin to the fibers distributed to the skin, viscera and muscles. Large somatic and large splanchnic fibers have origin from cells of the same type, the large clear cell; small somatic and small splanchnic fibers also from cells of the same type, the obscure cell. (5) Afferent fibers to the thoracic viscera arise from the first five dorsal ganglia. The number of such fibers in one instance was found to be 136 in the first dorsal, 192 in the second white ramus. (6) The smallest cells in the ganglion, under 26 mikrons in diameter, whether belonging to the obscure or clear type, are immature cells, and have no fully developed axon. (7) The coarsely granular cell is highly resistant to the axonal reaction, and may be a "relay" cell, but sufficient evidence it not forthcoming as to its significance. (8) The cells in the early embryo have the appearance of the smallest type described in the text. In the newly-born human subject the types present in the adult can be recognized. The morphological characteristics of the cells of early embryonic life are also met with in the reactive phase.

2. *Certain Tremors in Organic Cerebral Lesions.*—The author first offers, as a substitute for the careless use of the word *tremor*, the following definition: "A clinical phenomenon consisting in the involuntary oscillation of any part of the body around any plane, such oscillations being either regular or irregular in rate and in amplitude, and due to the alternate action of groups of muscles and their antagonists." Reports are presented and discussed of nine cases in which tremor was a constant symptom, the rate being slow, varying from 3 to 5 oscillations a second, in all cases more or less regular in rate when limited to one group of muscles, in some cases absolutely so; and either regular or irregular in the range of amplitude. Some cases showed a periodical rhythmical increase and decrease of the range. In some instances the tremor showed a remarkable tendency to persist, apparently indefinitely, limited to the one movement if the position of the limb and other conditions remained unaltered, whereas in other cases it would involve various groups of muscles for short periods in succession, or even simultaneously, the resulting movement of the limbs being in the latter case compound and irregular, as the various oscillations were not synchronous in rate or equal in range. In every case the patient was unable to check the movement except for the shortest space of time, and it was often increased by inhibitory attempts. In addition to the tremor proper, volitional movement of the limb affected was in every case complicated by an irregularity of the intention-tremor type. The tremor never persisted during sleep, nor when the limb was at rest with every segment supported separately. An excited psychic state or forcible movements of the opposite limb had the effect of increasing the tremor. Other symptoms, not invariably present but associated with the symptom-complex, were: rigidity, paresis and uselessness of the limbs, affection of coördination and an emotional state. The rigidity was slight, rather a stiffness perhaps, never associated with organic contractures, constant throughout a movement, and associated with a marked slowness in performing and especially in initiating voluntary movement. The weakness seemed to be largely subjective, and best described by the term uselessness. The peculiar emotional states comprised a tendency to laugh or cry uncontrollably without adequate cause, dulness and apathy, sometimes accompanied by paresis of the expressional movements of the face. Other symptoms merely coincident with the symptom-complex are of interest as indicating the localization of the various lesions. In eight of the cases ocular palsies were a marked symptom; loss or defect of the vertical movements was frequently noted. Other symptoms to be grouped with these were a gait resembling that of paralysis agitans or cerebellar disease, slight hemianesthesia not of the cortical type, hemiplegic weakness with increased reflexes and extensor plantar response, and in two cases homonymous hemianopia on the side of the affected limbs. From a consideration of these symptoms the author reaches the conclusion that in each of the nine cases the focus of disease involved the dorsal portion of the mid-brain, and argues that involvement of the nucleus ruber, or at least of the cerebello-rubro-spinal system, is the essential factor in the lesion, and that destruction rather than irritation of these parts is responsible for the tremor. Consequently, an inhibitory influence on cortical action must be assumed for the cerebello-rubral system, whether normally exerted on the higher or lower motor centers, on the cortex or the spinal cord, the author does not find enough evidence to decide. Literature shows little attempt to classify the various disorders of movement, but the evident difference between the varieties would justify rigidly separating them in clinical study and assuming that they must be due to or constantly associated with lesions different in nature and site. From a study of sixty cases with autopsy collected from the literature the author concludes that athetosis almost always results from disease of the basal ganglia, but especially of the optic thalamus; that choreiform movements occur with about equal frequency in lesions of the

basal ganglia and of the mid-brain, whereas in the large majority of the cases of tremor the lesion involves the mid-brain.

3. *Pathology of Infantile Paralysis*.—The author notes the three views held as to the pathology of acute anterior poliomyelitis, namely: (1) That the condition is due to direct affection of the anterior horn cells; (2) that the condition is due to acute inflammation in the region of the anterior horns; (3) that the condition is due to a vascular lesion, which may be either thrombotic or embolic. Cases do occur belonging to the first group, but do not clinically conform to the usual type of the disease. The second view is the one most commonly held, but it is the third to which the author subscribes, and which he supports by the report and discussion of three cases. Head and Campbell have recently argued that the thrombosis is secondary to an acute infection, probably of bacterial origin, because experimental occlusion of spinal cord vessels is not followed by inflammatory change or hemorrhage. Batten answers with a reminder that obliteration of a large vessel is not necessarily comparable to the condition produced by thrombosis occurring in a smaller vessel, and recalls the experiments of Prevost and Cotard, who, by means of fine tobacco seeds, produced infarcts in various organs, and proved that congestion, hemorrhage and the exudation of cells were the early results of obliteration of small vessels. The fact that the area of softening corresponds so closely to the distribution of the branch of the anterior main artery makes the suggestion a reasonable one that such a condition is due to an occlusion of a vessel or vessels. Furthermore, we find the disease occurring most frequently in the lumbar region, a fact to be accounted for by remembering that the blood supply of the cord is at a point most distal from the heart, and that the reinforcing arteries have a very long course.

4. *So-called Facial Hemihypertrophy*.—The author reviews pretty thoroughly the literature on this rare condition, including reports of some thirty cases besides his own, that of a girl of ten suffering from acquired hypertrophy of the left side of face and skull. The child was normal at birth, and the deformity was first noticed at six years of age as a bony enlargement on the head near the vertex, followed by a similar one in the occipital region. Then came a fulness in the forehead, and lastly the cheek began to enlarge. The facial enlargement is limited strictly by the median line, and includes all the soft parts and the facial bones, together with most of the cranial bones. There is marked bossing of the frontal, parietal and occipital bones. The molars are longer and thicker on the left side, and the mastoid, malar and zygoma show enlargement. The posterior portion of the skull is also enlarged, and the hair covering it coarser than on the right. Tongue, palate and tonsils normal. The right eye has normal vision, but in the left the field is contracted to the nasal side. Hearing normal on right, markedly defective on left. Special senses otherwise normal. Sensibility to touch, pain and temperature unaffected. The author then gives a digest of the clinical features of the cases reported, followed by a discussion of the pathology and pathogeny. He thinks it not possible, either on clinical or biological grounds, to maintain the distinction indicated by the words *congenital* and *acquired*, and would prefer to consider the morbid conditions in both classes to be the outcome of some disease process common to both, commencing prenatally in one class and postnatally in the other. Post-mortem observations of the condition are practically non-existent. Portions of soft tissue removed during life show overgrowth of sebaceous and sudoriparous glands, and of adipose and connective tissues, with numerous degenerated epithelial cells. The views on the pathogeny of the condition are many and varied. Many writers take that of vasomotor paralysis, others that of circulatory stasis in the affected part due to vicious positions in the uterus, a theory which leaves the postnatal cases quite unaccounted for. Some attribute it to stimulation or irritation of epiphyseal cartilage and osteoplastic periosteum. A teratological theory has

been advanced, but this again fails to account for the postnatal cases. Finally, the author is inclined to favor the theory first advocated by Lewin, who gave a trophic rather than a vasomotor explanation of local atrophies and hypertrophies. The occurrence at times of hypertrophic dystrophies in syringomyelia and acromegaly add color to this theory. His own case is the only one in which occipital bossing has been noted, and this might be interpreted as pointing to a possible connection with acromegaly. The author thinks the following conclusions are warranted: (1) While nothing is known of the pathogeny of racial hemihypertrophy, the most reasonable explanation of the condition is that which associates it with disturbance of hypothetical nutritional or trophic centers in the perpendymal gray matter of the encephalon. (2) In some of its features facial hemihypertrophy appears to have relationship with acromegaly and syringomyelia.

JELLIFFE.

PEDIATRICS.

(Vol. 16, 1904.)

1. Early Symptoms of Dementia Præcox. A. R. DIEFENDORF.
2. Cerebral Palsy of Childhood. M. L. GRAVES.
3. Natal Right Hemiplegia.
4. A Case of Hemiplegia Occurring During Whooping Cough and Diphtheria. HENRY FRASER.
5. Intraspinal Injections of Lysol in the Treatment of Cerebrospinal Meningitis. MORRIS MANGES.
6. Clinical Features of Cerebrospinal Meningitis of the Epidemic Type. HENRY KOPLIK.

1. *Early Symptoms of Dementia Præcox*.—Author speaks of the necessity of early recognition by the general practitioner. For convenience, groups the initial symptoms into four classes, namely, cases in which there is simple mental deterioration without hallucination or delusions; cases in which a few indefinite delusions appear; cases of a neurasthenic character; cases of hysteroid type.

2. *Cerebral Palsy of Childhood*.—Postnatal left hemiplegia. Occurred at eight years of age, during an attack of typhoid. At twenty years admitted to hospital because of mental impairment. No history of convulsive seizures.

3. *Natal Right Hemiplegia*.—Tedious birth, no instruments used. Fretful immediately following birth. Paralysis of right side noticed at three months of age. Epileptic convulsions at twenty months. At eight years mind normal; fair scholar. Abandoned school attendance because of frequency of convulsions. At sixteen committed to hospital because of impaired mental state. At nineteen general condition grave, mind nearly extinct.

4. *A Case of Hemiplegia Occurring During Whooping Cough and Diphtheria*.—Male child of eighteen months, ill with diphtheria and whooping cough. Antitoxin used. On sixth day of diphtheria had convulsion, followed by paralysis of right side. Right hemianesthesia. Later, convulsions of Jacksonian type, right lateral hemianopsia. Hemiplegia thought to be due to intracranial hemorrhage.

5. *Intraspinal Infections of Lysol in the Treatment of Cerebrospinal Meningitis*.—Three cases reported successfully treated by lumbar puncture and lysol injections, after a modification of the method of Seager, of Lisbon.

6. *Clinical Features of Cerebrospinal Meningitis, or Cerebrospinal Fever of the Epidemic Type*.—Paper very interesting, complete as to detail, etc. The disease, like pneumonia, considered to be self-limited. At present the most to be expected of treatment is to relieve suffering and prevent complications. Lumbar puncture cannot be considered as curative, although many consider it a procedure attended with decided benefit. Thirty-one cases treated by Seager (lumbar puncture and lysol injection) show a